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How Emotions Are Shaped by Bodily States

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Abstract

The state of the body is central to guiding motivational behaviours. Here we discuss how afferent information from face and viscera influence the processing and communication of emotional states. We highlight (a) the fine-grained impact that facial muscular and patterned visceral responses exert on emotional appraisal and communicative signals; (b) short-term changes in visceral state that bias brain responses to emotive stimuli; (c) the commonality of brain pathways and substrates mediating short- and long-term bodily effects on emotional processes; (d) how topographically distinct representations of different bodily states are coupled to reported feelings associated with subtypes of disgust; and (e) how pupil signals contribute to affective exchange. Integrating these observations enriches our understanding of emotional processes and psychopathology.

Keywords
autonomic, body, emotion, feelings, interoception, viscera

Bodily States and Emotional Feelings

A goal of cognitive neuroscience is to understand how the conscious mind is produced by, and influences, biological processes (Rees & Seth, 2010). Scientists and clinicians typically hold that subjective mental experience, thoughts, and feelings are hosted by the brain through its intrinsic neural activity. Other bodily organs, while under the brain's control, are not generally considered to have this relationship to consciousness. This distinction can therefore be exploited as a model for characterizing the physical correlates of mind through tracking neurally mediated interaction between changes in body (outside the brain) and changes in emotion and cognition. Body–mind interactions are of particular interest to understanding emotions and motivations. Bodily states shape mental content: If we are dehydrated we experience thirst, if nutritionally deprived, hunger. These basic motivational changes influence how we interact mentally and physically with our environment. Motivational changes are elicited by other external threats to the physiological integrity and by external stimuli that may ensure the body's integrity. Changes in the physiological state of the body accompany the processing of such aversive and appetitive motivational cues to facilitate and anticipate appropriate behaviours. Thus, stimuli with potentially negative or positive consequences become "emotionally-competent" (Damasio, 2001) and divert physiological resources to meet evolutionary imperatives (Panksepp, 1998). Arguably, if a stimulus elicits no change in bodily physiology then it cannot be viewed as emotive. Action programmes (stereotypical changes in bodily physiology) are evoked automatically by strongly salient stimuli without the initial need for cognitive appraisal. James (1994) and Lange (1885) proposed that the *emotion* follows the physiological response and is the feeling of that response. James used a number of analogies to describe what he meant by emotion; for example, proposing that the absence of this bodily response would result in a perception being "pale, colorless, destitute of emotional warmth." Walter Cannon challenged this view by arguing that the same emotion can occur in different bodily states, and that the differentiation of physiological arousal was too coarse to account for the richness of emotional experience (Cannon, 1927). A partial resolution was proposed by Schachter and Singer (1962), who argued that changes in the body can initiate and intensify emotions but must be interpreted cognitively to determine which emotion is experienced. In Schachter

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and Singer's (1962) experiment, participants were injected with saline or adrenaline and placed in a room with a "stooge" who acted irritable or happy; participants who were physiologically aroused by the adrenaline reported experiencing more emotion, the quality of which corresponded to that expressed by the stooge. This two-stage model allowed for complex emotional experience to be grounded on the interpretation of an undifferentiated emotional arousal state.

Over the last 20 years, the notion that emotions have a peripheral origin was reinvigorated by Damasio, Tranel, and Damasio (1991). Their "somatic marker hypothesis" developed from the observation that behavioural learning of the motivational significance of stimuli is coupled to physiological arousal responses evoked by the stimuli. An interpretation was that arousal responses reflect learning and (implicitly/preconsciously) guide decision-making. This interrelationship is lost in people with ventromedial prefrontal lesions who consequently fail to learn by experience despite retaining insight into what, in fact, would be appropriate behaviour (Damasio et al., 1991). Both emotion and cognition were argued to be subject to bodily biases (Damasio, 1994, 1999). Damasio's recent writings emphasize the primacy of emotion action programmes (triggered within brainstem regions such as the periaqueductal grey matter [PAG]) that may provide feedback and emerge as perceived feelings (Damasio, 2010). It is noteworthy that the ventromedial prefrontal cortical and adjacent subgenual brain area identified by Damasio's group as mediating the coupling of somatic state to motivational behaviours is functionally compromised in anhedonic and depressive states (Drevets et al., 1997; Keedwell, Andrew, Williams, Brammer, & Phillips, 2005; Mayberg et al., 2000; Mayberg et al., 2005), conditions that are associated with abnormalities in physiological reactivity (Carney, Freedland, & Veith, 2005).

Muscles as Context

Widely used tools used in emotion science are the Ekman emotional face stimuli (Ekman, 1972; Ekman & Friesen, 1978), depicting six "prototypical" emotions. These expressions originate from evolutionary-selected reactions (Darwin, 1998), are recognized in the same way across different human societies, and appear to be a "universal" means of exchanging core emotions among healthy humans (Ekman, 1972). Thus, individual Ekman stimuli are frequently used as experimental probes to evoke responses (at neural and behavioral levels) within brain systems supporting those specific basic emotions. This is interesting and potentially counterintuitive (e.g., fear responses are engaged more by processing fearful face stimuli than by threatening/anger stimuli). One interpretation is that when we see an emotion, we simulate the same state within our own brains and bodily states (including facial expression); so fear in others generates fear in us. However, recent human emotional neuroscience has focused on facial expressions at the expense of other responses (though Darwin, James, and Lange drew attention to the whole body's role in emotion). Gait and posture betray emotion at distances well before one can see salient facial features such the whites of eyes. Interactions between bodily and facial expressions of emotion have been highlighted by de Gelder et al. (2006). They show Ekman expressions are influenced by context. Thus, if an angry expression is placed on a "fearful" body then recognition accuracy drops by 20%. Clearly, there are competing signals within such hybrid stimuli. Part of that competition is at the level of embodied context; if a stimulus triggers a corresponding emotional bodily state in the viewer, this compromises the ability to judge the facial emotion.

The role of feedback from one's own body state as a context for appraising emotional material was elegantly illustrated behaviourally by Strack, Martin, and Stepper (1988), who focused on facial expressions. Participants judged how funny cartoons were, while holding a pen with either their teeth or lips. These manipulations interfered with evoked emotional facial muscle responses: You cannot smile if you hold a pen with your lips. You activate the same muscles as smiling if you hold the pen with your teeth. A cover story ensured participants were unaware that these were "emotional" manipulations, yet cartoons were perceived as funnier if the pen was held with the teeth and less funny if held in the "pouting" lip position. This finding, among others, led to the theoretical formulation of the facial readout (or feedback) hypothesis, which, at its most "Jamesian" extreme, suggests that we might experience things as funny or pleasant because we feel ourselves smile (James, 1994). Emotional facial reactions can be automatic: Valenced emotional stimuli elicit covert electromyographic (EMG) responses in the "smile muscle" (zygomaticus) or "frown muscle" (corrugator supercilli) even to subliminal stimuli (i.e., for which the viewer has no conscious recollection of having processed them; Dimberg, Thunberg, & Elmehed, 2000; Sonnby-Borgström, 2002). Facial muscles develop from branchial arches (i.e., origins of the gill system of fishes) and facial nuclei are proximal to autonomic control centres. The implication is that facial expressions are similar to, and intrinsically coupled with, unconsciously regulated visceral arousal states (see Porges, 1997).

Automaticity in facial expressions is evident in expression contagion. High-speed cameras can surreptitiously film people watching another's facial expression to show subtle mimicry in the facial movements of the observer (similar to EMG responses observed by Dimberg et al., 2000). Contagion provides, through facial feedback, a mechanism for representing how others feel by using the data coming back from our own automatically reacting faces. In our lab, we explored this experimentally: Participants were cued to smile or frown after viewing neutral, smiling, or frowning face stimuli. Using EMG, we showed that it was harder to smile quickly if you saw a frowning expression and harder to frown if watching a smile. Using functional magnetic resonance brain imaging (fMRI), we located corresponding changes in brain activity in a set of areas that linked the processing of facial expressions (superior temporal sulcus; STS), internal emotional feeling (insula), and effort (cingulate). Activity in these areas also reflected differences across individuals in measures of empathy (Lee, Dolan, & Critchley, 2008). In another experiment closer to that of Strack and

colleagues (1988), we asked people to smile, frown, or hold neutral expressions while they rated emotional face stimuli (Lee, Dolan, & Critchley, 2011). If the participant was frowning, this reduced their perception of happiness in smiling faces and enhanced their perception of anger (by approximately 6%), while if they smiled, frowning faces appeared milder and smiling faces happier. In the brain, these effects occurred within the amygdala (a rapid detector for emotional salience) and the insula (implicated as the readout for internal bodily feeding states). Together these observations highlight musculature self-reference in shaping emotional brain processes and responses, but raise an important issue regarding the boundaries between self and other; in the phenomenon of contagion, the emotions are typically attributed to another.

Mechanisms underlying motoric simulation of emotions can be compromised in affective disorders and specific neurological conditions. The negative underreactive expression of a depressed person can exacerbate that individual's disengagement from interactions with other people who may find the experience unrewarding and depressing. For the depressed individuals themselves the fixed depressive facial expression can negatively bias stimulus processing and encoding into memory. While the causality is complex in depression, neurological conditions that compromise facial expression including Parkinsonism, Moebius syndrome, and even the cosmetic use of botulinuum toxin have been reportedly associated with flattening of subjective feelings and vulnerability to depressive mood disorder (Davis, Senghas, Brandt, & Ochsner, 2010; Gillberg & Steffenburg, 1989; Katsikitis & Pilowsky, 1991). In contrast, enhanced emotional contagion of facial expression accompanies Tourette syndrome (and perhaps attention deficit hyperactivity disorder [ADHD] and hypomania), where there is a link to emotional lability and instability (e.g., Cavanna, Robertson, & Critchley, 2008).

Perturbation of Internal Visceral State

The internal state of the body, in health, is closely regulated through neural (autonomic) and humoral control systems in a way that signals motivational needs, facilitates adaptive behaviours, and permits stimulatory understanding of others' emotion. Sickness illustrates how perturbed internal bodily state can change thoughts and feelings: If you are "under the weather" because of an infection, mood drops, reaction times slow, you fatigue easily, and thoughts get muddled. This is an expression of a stereotyped specific response to inflammation that directs bodily defences toward recuperation (Dantzer & Kelley, 2007). Neuroimaging research has addressed underlying mechanisms: Inducing a mild transient inflammatory response with typhoid vaccination (for overseas travel) will evoke increases in circulating immune chemicals such as cytokines. Mood will drop within 4 hours and self-reported fatigue and confusion increase (recovering within 24 hours). Neural responses change: Alterations in subgenual cingulate reactivity correlate with the degree of inflammation-evoked depression, while other related regions predict the impact of inflammation on cognition and fatigue (dorsal cingulate cortex, insula) (Harrison, Brydon, Walker, Gray, Steptoe, & Critchley, 2009; Harrison, Brydon, Walker, Gray, Steptoe, Dolan et al., 2009). Changes in subgenual cingulate cortex are of particular interest as the region is linked to anhedonia, depression, and response to antidepressants (Mayberg et al., 2000). It is also a target for deep brain stimulation for treatment-resistant depression (Mayberg et al., 2005). The vegetative and psychological effects of inflammation-induced sickness suggest that clinical depression may represent aberrant engagement of adaptive tonic reactions to visceral perturbation. Here effects are sustained over time, but in principle the same psychophysiological mechanisms may operate over much shorter periods.

Short-Term Autonomic Fluctuation and Central Processing

Dynamic fluctuations in heart rate and blood pressure interact with emotional processing and experience (Critchley, 2009). The contextual expression of these effects is apparent in studies of patients with peripheral autonomic denervation who cannot control their cardiovascular responses (Critchley, 2009; Critchley, Mathias, & Dolan, 2001, 2002; Critchley, Nagai, Gray, & Mathias, 2011). However, rapid changes in cardiovascular arousal also impact on our feelings and thoughts. A central aspect of cardiovascular autonomic control is the regulation of blood pressure to meet, among other things, postural demands (e.g., stopping us from fainting when we stand). Changes in blood pressure are sensed through baroreceptors (sensors in the large arteries), which fire with each heartbeat indicating arterial pulse pressure (Gilbey, 2007; Jänig, 2006). This signalling contributes to the baroreflex; blood vessels dilate and heart rate slows if pressure is too high, or conversely vessels constrict and heart rate quickens if the pressure is low. Baroreceptor activation provides a major interoceptive route to the brain (the representation of which may emerge as feelings of physical arousal). This baroreceptor signal is phasic and pulsatile, suggesting that whatever cognitive/emotional processes are sensitive to cardiovascular arousal may have intrinsic short-term rhythmicity.

Experimentally, salient stimuli such as electric shocks or faces can be briefly presented at different phases of the cardiac cycle, either when the heart has just ejected blood (during natural baroreceptor activation) or when it is firing (between bursts of baroreceptor activity). Combining autonomic monitoring with neuroimaging reveals some interesting effects: Automatic blood pressure reactions to electric stimulation to the skin are blocked by natural baroreceptor activation when shocks are delivered at systole. This interaction between internal signals and stimulus processing is associated with changes in neural responses of amygdala, insula, and dorsal brainstem which also mediate contextual effects of individual differences between participants in autonomic parasympathetic tone (Gray, Rylander, Harrison, Wallin, & Critchley, 2009). Thus, our processing of salient environmental stimuli can fluctuate within each heartbeat interval. Such short-term changes in bodily context (baroreceptor activation) selectively affect conscious appraisal of stimuli, rather than "low-level" sensory detection. We have now shown this for electroencephalography (EEG) response to pain (Gray,

Minati, Paoletti, & Critchley, 2010) and for detection of flicker in visual stimuli (unpublished observations). If we look at emotional face stimuli, baroreceptor activation alters some emotional expressions (e.g., disgust and happiness) but not others (sadness and anger) (Gray et al., 2011), implying that the effect is coupled to parasympathetic control (more prominent in happiness and disgust). Together short-term phasic changes in cardiovascular state modulate emotional judgements, and behavioural/autonomic responses to motivationally salient stimuli. The brain mechanisms involved, dorsal brainstem, insula, and amygdala, appear the same as those also sensitive to tonic perturbations of bodily state.

Distinct Visceral States and Affective Exchange

The James–Lange theory implies that different internal bodily states engender different emotions. The evidence for this was limited to only a few studies that link emotional experience to patterning across different axes of autonomic response (e.g., Ekman, Levenson, & Friesen, 1983; Kreibig, 2010; Rainville, Bechara, Naqvi, & Damasio, 2006). Thus, the concept that there are multiple bodily arousal states is not widely embraced in psychology, yet autonomic neuroscience highlights the organ-specific autonomic control of peripheral arousal states consistent with cultural and experiential associations linking emotions with distinct visceral feelings; for example, the heart skipping a beat with fear, the face going red with anger.

An examination of disgust representation provides a clearer picture of the relationship between central emotional experience and internal physiological responses. The term disgust is used as a label for different types of negative emotional experiences, from visceral reactions to contaminated food to rejection of social immorality. Even at its most basic, there is more than one sort of disgust: The type that makes you vomit (usually involving contamination and food) and the type that makes you faint ("body boundary violation" [BBV] disgust, usually involving active breaching of the skin and the presence of blood), indicating physiological differentiation within a single verbal descriptor. In an fMRI study, participants watched video stimuli designed to elicit degrees of "nauseating" and "bloody" disgust while brain activity was measured concurrently with electrical responses of the stomach and the heart (Harrison, Gray, Gianaros, & Critchley, 2010). The responses of the stomach to food-related (nauseating) scenes evoked focal activity within bilateral regions of insula cortex and this neural activity also predicted how strong the participant rated the experience of disgust. The response of the heart to surgical/bloody stimuli (BBV disgust; withdrawal of parasympathetic cardiac influences) evoked activity within a different subregion of left insula cortex, which again predicted subjective ratings of disgust feelings. This study is perhaps the first to illustrate direct neural coupling (i.e., through shared regional brain substrates) of bodily state with emotional feeling state. Insula cortex is more generally implicated as a substrate for declarative feelings originating in internal physiological changes; for example, states of cardiorespiratory arousal linked to anxiety states (Craig, 2004; Critchley, Wiens, Rotshtein, Ohman, & Dolan, 2004). There are strong relationships between disgust and psychopathology. Disgust is often intrinsic to the symptomatology of mood and anxiety disorders. Contamination fears feature often in obsessive-compulsive disorder and major depression—in the latter this can have strongly self-directed and moral content. Predisposition to disgust-triggered autonomic responses is linked to vulnerability to anxiety states (Olatunji, Cisler, McKay, & Phillips, 2010). People who faint at the sight of blood are twice as likely to develop an anxiety disorder as people without a personal history of fainting (Kouakam et al., 2002).

Pupillary Response and Affective Exchange

The methods used to probe disgust responses tap into vicarious aspects of emotion including facial signals (as discussed before). Some visceral correlates of emotion are overtly communicative, for example, blushing or pallor. Other signals, like changes in pupil size, are more subtle, yet these automatic unconscious processes still betray our fears and feelings to others. Schachter and Singer's (1962) model of emotion suggests that increases in arousal trigger and intensify emotion, but that the quality of the emotion is cognitively interpreted from other contextual cues. One signal for physiological arousal (heuristically increased sympathetic decreased parasympathetic tone) is pupil dilatation. Pupils dilate when one is more aroused. A few years back, we wondered what effect pupil size might have on the processing of different emotional facial expressions. We altered pictures of emotional faces to portray different sizes of pupils. We predicted that bigger pupils would intensify the rated intensity of all the different emotions; we were wrong. Behavioral tests, repeated on a number of occasions, showed pupil size affected only the judgement of sad faces; smaller pupils on sad faces made the face look more negative and more intensely sad to healthy raters (who were not explicitly aware of the pupil manipulation). People who were more sensitive to this covert pupil effect scored higher on an empathy questionnaire. We scanned people looking at these face stimuli and showed that brain areas implicated in emotion perception (amygdala, insula, STS) were specifically engaged (Harrison, Singer, Rotshtein, Dolan, & Critchley, 2006; Harrison, Wilson, & Critchley, 2007). The next finding was also surprising: During scanning, we measured the observers' own pupil size and observed a mirroring of the pupil response only when viewing sad faces. We had expected that pupil size would either have no effect, or that it would be copied automatically irrespective of what emotion was portrayed. Yet if a participant saw a small pupil on a sad face his/her own pupil constricted, and this was not true for other emotional expressions. The degree to which people mirrored the pupil in sadness also predicted their empathy scores. Within the brain, this effect was expressed in the activity of a dorsal brainstem region that contained the (Edinger-Westphal) nuclei controlling one's own pupil (activity within this same region also correlated with the degree of contagion of pupils) (Harrison et al., 2006). This study highlights the contribution of pupillary signals in communicating emotional states and

the embodiment (simulation/mimicking/contagion) of those responses in the viewer in a way that follows the individual empathetic responses, but which may also directly influence judgements of emotion (either because dilated pupils compromise visual accommodation, or because there is active feedback from the pupil muscle itself; a topic of ongoing experimental study). For pupils the effect is powerful for sadness and does not appear to generalize to other expressions of emotion.

A further neuroimaging study examined what happens in the brain when two people are engaged in dynamic eye-to-eye contact and experience either matching or unmatched changes in pupil size (Harrison, Gray, & Critchley, 2009). A stimulus (pair of eyes) was linked to the output of an eye tracker in the fMRI scanner. For some periods, when the viewers' pupils constricted, the stimulus pupils constricted, and for other periods, when the viewer's pupils constricted, the stimulus pupils dilated. We found the following: The more the stimulus pupils changed in size, the more activity was evoked within amygdala and STS. When pupils varied congruently (coupled in the same direction), there was little extra effect on brain activity. However, when the size of observed (stimulus) pupils changed in the opposite way to the pupils of the viewer, this powerfully activated brain region linked to emotional arousal (including anterior cingulate and insula cortices), indicating that pupil mismatch is an important cue to evoking activity within the "salience network" (Seeley et al., 2007). A speculative interpretation is that if you are talking to another person, pupils tend to harmonize, both because of ambient lighting and "bodily attunement" through shared emotionality. If there is an unexpected, unmirrored change in your companion's pupillary state of arousal, this breaks the harmonization and is a salient event signalled in the brain, which may be of sufficient magnitude to interrupt the flow of cognition and behavior and redirect attention toward the other's mental state. This effect during eye-to-eye contact is dependent on subconscious feedback from your own pupils against which the pupillary responses of another person are gauged. Clinically, the reactivity of pupils is compromised in depression. In sadness and pain, the mechanisms through which the pupils constrict can be uncoupled from the reactivity to ambient light.

General Conclusions

Bodily states act as a context for emotions, shaping affective processes in a manner similar to the effects of external contexts and occasion setters (Bouton, 2001). However, they do more, as they are a source of affective feeling that arises from highly patterned physiological states. The changing representation of internal bodily "self" exerts a dynamic contextual effect on emotional processing (even changing within a single heartbeat). The same basic mechanisms appear to underlie brain–body interaction across different timeframes from baroreceptor activation, emotion-evoked autonomic reactions, embodied mood states, and tonic interoceptive perturbations associated with illness and inflammation. These mechanisms also extend beyond the individual to the communication and exchange of emotional states and can be engaged to appraise the feelings of others. Neuroimaging in combination with physiological monitoring permits the dissection of these processes at the level of coordinated neural systems, competing processes, and modular substrates for experience and behavior. These basic approaches in healthy individuals allow us to address in a more comprehensive manner states of psychopathology in which constitutional differences in physiological reactivity or interoceptive sensitivity are linked to vulnerability to mood and anxiety disorders, the presence of daily mood and anxiety symptoms, and fluidity of social interaction (Fukushima, Terasawa, & Umeda, 2011; Hubert, Wicker, Monfardini, & Deruelle, 2009; Paulus & Stein, 2010). At a deeper level it has been argued that viscerosensory mechanisms are central to integrity of self (Damasio, 2010). Individual differences in interoceptive awareness not only predict sensitivity to the occurrence of mood symptoms (Critchley et al., 2004; Paulus & Stein, 2010), but also susceptibility to illusory distortions in self-representation (Tsakiris, Tajadura-Jiménez, & Costantini, 2011). Dysfunctional processing of the mismatch between predicted and actual internal bodily states (prediction errors) has been proposed to underlie anxiety disorder (Paulus & Stein, 2010) and psychosis (Palaniyappan & Liddle, 2011). Research into the control of bodily states provides a grounded approach to the neurobiology of subjective experience, without which understanding of psychiatric illness would be impoverished.

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